cannot be confirmed as a higher frequency of conduction abnormalities in arrhythmogenic cardiomyopathy can be definitely excluded. These findings correspond to fibrofatty lesions in Tabib's work possibly representing true conduction abnormalities within arrhythmogenic cardiomyopathy usually sparing the conduction system.

References

[1] Tabib A, Loire R, Chalabreysse L, et al. Circumstances of death and gross and microscopic observations in a series of 200 cases of sudden death associated with arrhythmogenic right ventricular cardiomyopathy and/or dysplasia. Circulation 2003;108:3000–5.
[2] Quarta G, Syrris P, Ashworth M, et al. Mutations in the Lamin A/C gene mimic arrhythmogenic right ventricular cardiomyopathy. Eur Heart J 2012;33:1128–36.
[3] Taylor M, Graw S, Sinagra G, et al. Genetic variation in titin in arrhythmogenic right ventricular cardiomyopathy-overlap syndromes. Circulation 2011;124:876–85.
[4] Marshall W, Furey M, Larsen B, et al. Correspondence to: right ventricular cardiomyopathy and sudden death in young people. N Eng J Med 1988;319:174–5.
[5] Marcus FI, McKenna WJ, Sherrill D, et al. Diagnosis of arrhythmogenic right ventricular cardiomyopathy/dysplasia: proposed modification of the Task Force Criteria. Eur Heart J 2010;31:806–14.
[6] Peters S, Trümmel M, Koehler B. Special features of right bundle branch block in patients with arrhythmogenic right ventricular cardfiomyopathy/dysplasia. Int J Cardiol 2012;157:102–3.
[7] Peters S, Trümmel M, Koehler B. QRS fragmentation in standard ECG as a diagnostic marker of arrhythmogenic right ventricular dysplasia–cardiomyopathy. Heart Rhythm 2008;5:1417–21.
[8] Vasawala SC, Finn C, Delpriore J, et al. Prospective study of cardiac sarcoid mimicking arrhythmogenic right ventricular dysplasia. J Cardiovasc Electrophysiol 2009;20:473–6.
[9] Martini B, Nava A, Thiene G, et al. Ventricular fibrillation without apparent heart disease: description of six cases. Am Heart J 1989;118:1203–9.
[10] Hodgkinson KA, Parfrey PS, Bassett AS, et al. The impact of implantable cardioverter-defibrillator therapy on survival in autosomal-dominant arrhythmogenic right ventricular cardiomyopathy (ARVD5). J Am Coll Cardiol 2005;45:400–8.
[11] Von Tintelen JP, Van Gelder IC, Asimaki A, et al. Severe cardiac phenotype with right ventricular predominance in a large cohort of patients with a single missence mutation in the DES gene. Heart Rhythm 2009;6:1574–83.

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Long-term prognostic value of preprocedural adiponectin levels in patients undergoing percutaneous coronary intervention☆

Cédric Delhaye [a,*], Natalia Kpogbemabou [a,b], Thomas Modine [a], Gilles Lemesle [a], Bart Staels [c], Michael Mahmoudi [d], Anne Tailleux [c], Gérald Luc [e], Christophe Bauters [a,b], Jean-Marc Lablanche [a,b], Arnaud Sudre [a]

[a] Hôpital Cardiologique, CHRU de Lille, boulevard Pr Leclercq, 59037 Lille cedex, France
[b] Faculté de médecine, Université de Lille 2, 59045 Lille, France
[c] Inserm, U1011, F-59000, Lille, France; Université Lille 2, F-59000, Lille, France; Institut Pasteur de Lille, F-59019, Lille, France; European Genomic Institute for Diabetes (EGID), FR 3508, F-59000 Lille, France
[d] University of Surrey, Guilford, Surrey, GU2-7XH, UK
[e] EA 2694, Laboratoire de Santé Publique, 59045 Lille cedex; Faculté des Sciences Pharmaceutiques et Biologiques, Université de Lille 2, 59006 Lille, France

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Adiponectin, an adipocyte-derived protein, is considered to possess cardioprotective properties mainly through anti-inflammatory and anti-atherogenic mechanisms [1]. Low levels of adiponectin have thus been suspected to be associated with an increased risk of coronary artery disease (CAD) and acute coronary syndrome (ACS) in several though not all studies [2]. In patients presenting with manifest CAD, an inverse association has been suggested; high rather than low adiponectin levels may be associated with adverse outcomes and death [3–6]. In those presenting with acute ST elevation myocardial infarction (STEMI), adiponectin has been shown to be a predictor of all cause mortality or cardiovascular death at long follow-up [5]. However, there is currently limited data regarding the long-term prognostic value of adiponectin in patients undergoing percutaneous coronary intervention (PCI) outside the setting of STEMI. Furthermore, the relationship between adiponectin and stent-related complication such as restenosis has not been explored. This study was design to examine these issues.

Between March 2006 and September 2007, we constructed a prospective cardiovascular registry of 477 patients who underwent PCI for stable angina and non-ST-elevation ACS and for whom total plasma adiponectin were systematically assessed at the time of the procedure. Patients presenting with a STEMI were not included. Each patient gave informed consent. This study has received authorization from the local Patient Protection Committee. The authors of this manuscript certified that they comply with the Principles of Ethical Publishing in the International Journal of Cardiology.

Circulating levels of plasma total adiponectin were determined with a multiplex bioassay using commercially available kit from Linco Research Inc. (Billerica, MA, USA; Human cardiovascular disease panel 1 multiplex immunoassay). PCI with bare metal or drug-eluting stent (DES) implantation was performed using conventional techniques. Aspirin and clopidogrel were systematically used before the procedure and thereafter.

The median follow-up was 3.7 years [interquartile range = 3.4–4.2years]. The primary end point was defined as either all-

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* Corresponding author. Tel.: +33 320445301; fax: +33 320444898.
E-mail address: cedricdelhaye@yahoo.fr (C. Delhaye).

Table 1
Baseline characteristics stratified by adiponectin tertile.

	Total ($N = 477$)	Tertile1 ($n = 159$), Adipo < 15.25 μg/ml	Tertile2 ($n = 159$), 15.25 < Adipo < 20.12	Tertile3 ($n = 159$), Adipo > 20.12 μg/ml	r	*P*
Age (year)	62.5 ± 11	59.6 ± 10	63.1 ± 11	64.7 ± 11	0.20	<0.001
Male gender, *n* (%)	399 (83.6)	139 (87.4)	140 (88.1)	120 (75.5)		<0.001
Diabetes mellitus, *n* (%)[b]	148 (31.0)	53 (33.3)	49 (30.8)	46 (28.9)		0.607
Systemic hypertension, *n* (%)	280 (58.7)	93 (58.5)	92 (57.9)	95 (59.7)		0.696
Current smoker, *n* (%)	137 (28.7)	50 (31.4)	48 (30.2)	39 (24.5)		0.080
Hypercholesterolemia, *n* (%)[c]	395 (82.8)	138 (86.8)	131 (82.4)	126 (79.2)		0.010
Body mass index (kg/m^2)	27.6 ± 4.4	28.4 ± 4.1	27.8 ± 4.4	26.4 ± 4.5	−0.21	<0.001
Previous CAD, *n* (%)[d]	266 (55.8)	98 (61.6)	95 (59.7)	73 (45.9)		0.028
Indication for PCI, *n* (%)						
Stable CAD	316 (66.2)	107 (67.3)	110 (69.6)	99 (62.3)		0.492
Unstable angina	37 (7.8)	15 (9.4)	10 (6.3)	12 (7.5)		
Non-ST elevation MI	124 (26)	37 (23.3)	39 (24.5)	48 (30.2)		
Left ventricular ejection fraction, %	53 ± 10	54 ± 9	53 ± 11	53 ± 11	−0.03	0.561
Laboratory characteristics						
Baseline glucose (g/l)	1.02 ± 0.30	1.07 ± 0.34	1.02 ± 0.28	0.97 ± 0.26	−0.11	0.019
Serum creatinine (mg/l)	9.9 ± 0.3	9.3 ± 0.3	9.7 ± 0.2	10.8 ± 0.9	0.02	0.680
Total cholesterol (g/l)	1.49 ± 0.40	1.45 ± 0.41	1.53 ± 0.38	1.50 ± 0.41	0.04	0.351
LDL cholesterol (g/l)	0.87 ± 0.33	0.83 ± 0.34	0.89 ± 0.32	0.87 ± 0.34	0.01	0.846
HDL cholesterol (g/l)	0.39 ± 0.11	0.36 ± 0.09	0.41 ± 0.12	0.43 ± 0.12	0.32	<0.001
Triglycerides (g/l)	1.15 ± 0.70	1.24 ± 0.81	1.15 ± 0.54	0.95 ± 0.44	−0.20	<0.001
BNP (pg/ml)[a]	117 [25–128]	85.02 [20–87]	119.48 [30–128]	146.03 [30–179]	0.20	<0.001
Hs-CRP (mg/l)[a]	2.00 [0.74–5.88]	2.16 [0.88–5.57]	1.89 [0.74–6.25]	1.96 [0.66–5.93]	−0.04	0.427
Adiponectin (μg/ml)	17.22 ± 0.29	9.99 ± 0.29	17.78 ± 0.10	23.90 ± 0.25		
Angiographic characteristics						
Multivessel CAD, *n* (%)	359 (75.3)	125 (78.6)	118 (74.2)	116 (73.0)		0.244
Target coronary vessel: Left main or Left anterior descending, *n* (%)	182 (38.2)	61 (38.4)	57 (35.8)	64 (40.3)		0.761
ACC/AHA type C lesion, *n* (%)	108 (22.6)	41 (25.8)	35 (22.0)	32 (20.1)		0.108
Procedural characteristics						
Number of treated lesion per patient	1.24 ± 0.56	1.31 ± 0.59	1.26 ± 0.62	1.14 ± 0.45	−0.08	0.095
Drug eluting stent use, *n* (%)	350 (73.4)	130 (81.8)	114 (71.7)	106 (66.7)		0.003
Number of stent per patient, *n* (%)	1.36 ± 0.67	1.43 ± 0.68	1.42 ± 0.74	1.23 ± 0.56	−0.14	0.002
Glycoprotein IIb/IIIa inhibitors, *n* (%)	134 (29.1)	50 (32.9)	38 (25.0)	46 (29.3)		0.269
Bivaluridin, *n* (%)	133 (27.9)	44 (27.7)	51 (32.1)	38 (23.9)		0.895
Take home treatment prior to PCI, *n* (%)						
Aspirin	339 (71.1)	116 (73)	112 (70.4)	111 (69.8)		0.591
Thienopyridine	216 (45.3)	73 (45.9)	75 (47.2)	68 (42.8)		0.566
βBlocker	292 (61.2)	106 (66.7)	98 (61.6)	88 (55.3)		0.008
Angiotensin-converting enzyme inhibitors or receptor blocker	339 (71.1)	107 (67.3)	123 (77.4)	109 (68.6)		0.751
Statin	352 (73.8)	126 (79.2)	114 (71.7)	112 (70.4)		0.348

Data are expressed as mean ± SD or median [25–75th percentile]; *r*, correlation coefficient.
p values are for analyses performed with adiponectin as a continuous variable.
Abbreviations: ACC/AHA, American College of Cardiology/American Heart Association; BNP, brain natriuretic peptide; CAD, coronary artery disease; HDL, high-density lipoprotein; Hs-CRP, high-sensitivity C-reactive protein; LDL, low-density lipoprotein; MI, myocardial infarction; PCI, percutaneous coronary intervention.
[a] Analysis were performed using log transformed variable such as ln(BNP + 1) and ln(CRP).
[b] Defined as any history of diabetes mellitus and/or use of hypoglycemic drugs; also included new diagnosis made during index hospitalization with fasting glucose level ≥ 1.26 g/L on ≥ 2 different occasions.
[c] Included both patients with previously documented diagnosis of hypercholesterolemia treated with diet or medication; a new diagnosis could be made during hospitalization by elevated total cholesterol ≥ 160 mg/dl.
[d] Defined as prior to MI, coronary artery bypass graft or PCI.

cause mortality or MI or stroke. MI was defined as proposed by the guidelines [7]. Stroke was defined as the occurrence of a new neurological deficit and was confirmed by a neurologist and on imaging. Target vessel revascularization (TVR) was an examined secondary end point and characterized by ischemia-driven percutaneous or surgical revascularization of the treated vessel.

We categorized adiponectin into tertiles for the purposes of presentation. The relationships between adiponectin as continuous variable and other parameters were analysed by Spearman's correlation for continuous variable and by Student's *t* test for categorical variables. To study the relation between adiponectin level and multiple determinants, a multiple linear regression analysis with stepwise forward model was performed including potential explanatory variables having an association after univariate analysis ($p < 0.1$). For that, all variables in Table 1 were considered apart from procedural characteristics. Survival curves were constructed using the Kaplan–Meier method and differences between adiponectin tertiles tested by a log-rank test. Hazard ratios (HR) were calculated using univariate and multivariate Cox models. A stepwise forward procedure was used to determine independent predictors of events. Candidate covariates for multiple Cox regression analysis for death/MI/Stroke were chosen based on both the *p* value <0.10 in Cox univariate analysis (age, diabetes, left ventricular ejection fraction, baseline serum creatinine level, LDL,